# Dermal Microvasculature in Psoriasis

To the Editor:

Recently Braverman and Sibley described their observations regarding the ultrastructure of dermal capillaries and the labeling index of basal cells in psoriasis during different therapies [1]. The authors showed a lack of response of the dermal microvasculature to methotrexate and topical corticosteroids. During PUVA and Goeckerman treatment the same authors observed a prompt normalization of the dermal microvasculature before the labeling index of the basal cell layer reverted to normal [2].

They concluded that normalization of microvasculature is responsible for the longer remission periods during the latter two therapies and that the fast relapses following discontinuation of methotrexate and corticosteroids are the consequence of incomplete treatment of the dermal capillaries. This implies that the changes of dermal capillaries are of significance for the pathogenesis of psoriasis. We wish to congratulate Drs. Braverman and Sibley with this synthesis of ultramorphology, cell cycle kinetics, and clinical experience.

Some years ago the Nijmegen group examined the dynamic aspects of the psoriatic lesion: initiation (response to standardized injury), progression (the margin zone of spreading psoriatic lesions) and therapeutic regression (PUVA, topical corticosteroids). We measured epidermal cell cycle kinetics and some marker enzymes, including acid phosphatase (keratinization), glucose-6-phosphate dehydrogenase (epidermal proliferation), and alkaline phosphatase (capillaries). We reached the following conclusions:

A. Following standardized skin injury the capillaries of psoriatic patients overreact in comparison with the epidermal parameters [3].

B. In the margin zone of spreading psoriatic plaques the capillaries are abnormal up to 1.2 cm outside the clinical boundary, whereas epidermal abnormalities are seen only 0.4 cm outside the clinical edge [4].

C. During PUVA therapy the parameters of the epidermis and capillaries normalize at the same speed. By contrast, corticosteroids have no response on the marker for capillaries [5].

We now may reach a synthesis in which molecular biology takes part as well. The capillary changes are *not* an epiphenomenon in the pathogenesis of psoriasis, and normalization of capillaries is essential for sustained remissions.

## REFERENCES


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# Fibronectin Receptor Function

To the Editor:

We have several questions for Drs. Takashima and Grinnell, whose recent article concerning epidermal outgrowth from explants [1] we read with great interest and benefit.

A. Figure 1 is an experiment which shows that after 3 days, subsequent epidermal migration is fibronectin dependent. Do the authors have any information on whether fibronectin plays a role in the initiation of migration from days 0–3?

B. In the same experiment, 400 $\mu$g/ml of antifibronectin antibody or preimmune IgG was used. Is this not an excessive amount of protein to be adding? What is the approximate ratio of antifibronectin antibody molecules to fibronectin molecules? Could this large amount of protein be producing nonspecific effects?

C. Since epidermal outgrowth after day 3 can be due to epidermal cell mitosis [2], is there any information on whether fibronectin affects the mitotic phase of epidermal outgrowth from explants?

D. Figure 4 shows that by day 4, 80% of the keratinocytes in culture are basal cells. The authors hypothesize in the discussion that it is this increased pool of basal cells which exhibits fibronectin receptor function. However, Figure 3 shows that on day 4, approximately 5% of the cells attach. Do the authors think this is due to a lag in the expression of fibronectin receptor function?

Overall, we found the paper of great interest and believe the results to be of great importance in understanding epidermal wound healing.

## REFERENCES


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